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With some Considerations on its Pathologic Aspects.

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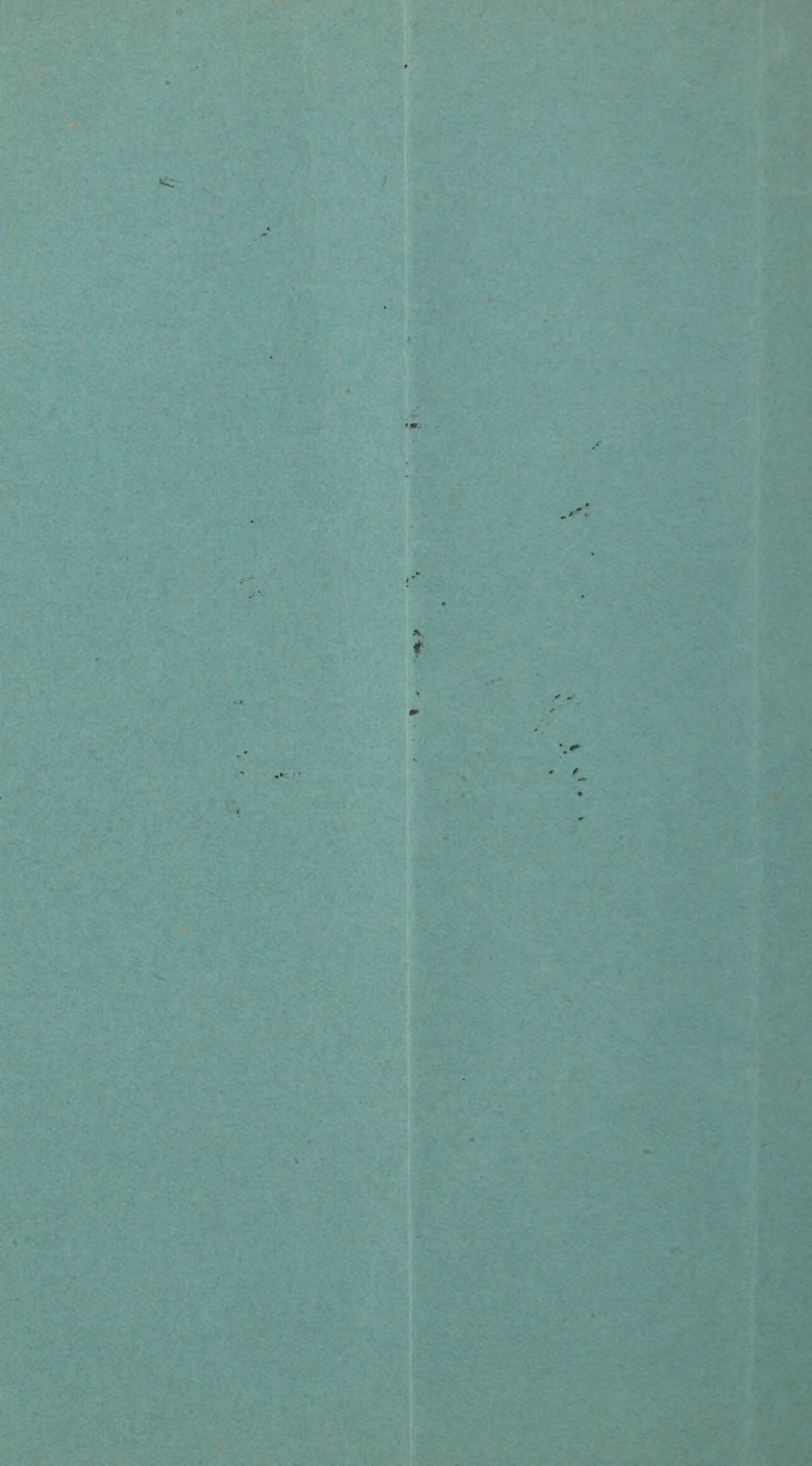
PATHOLOGIST AND SECOND ASSISTANT PHYSICIAN TO THE NEW JERSEY
STATE HOSPITAL AT MORRIS PLAINS.



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AUTHORITIES are at such variance as to the existence or non-existence of brain-lesions in acute delirious mania that I deem the following case of especial interest, presenting as it does both macroscopic and microscopic lesions of some import:

L. S., a colored farm-laborer, twenty-five years old, was admitted to the New Jersey State Hospital at Morris Plains, April 27, 1894, with the following history: The patient had always been regular in his habits and industrious. About one week before admission he suddenly manifested maniacal tendencies, and threatened his employer's life. The authorities immediately took him in custody, and after a sojourn of about one week in the county jail he was brought to this institution in restraint. The patient was so much exhausted that for a short while after admission he was very quiet; but he soon became maniacal, singing and shouting at the top of his voice, and pacing about the ward in an excited manner. He slept very little during his first night in the institution, and was considerably excited during the next day. He took a little food at dinner-time, and slept during the following night under the influence of paraldehyd. On the next morning he seemed very quiet, but listless, occasionally ejecting a quantity of greenish, frothy material from his stomach. Nausea



subsided under the influence of small doses of calomel frequently repeated, and the man slept fairly well during the night. On the following day it was determined to feed him, as he had up to this time taken very little food, and he seemed to be fast losing ground; he was accordingly given a sufficient quantity of milk both morning and evening by means of the nasal catheter. During the afternoon he was noisy, but slept fairly well during the night.

On May 1st the man had slight fever in the morning. The tongue was dry. The patient was very quiet all day, and seemed on the whole slightly better. He took milk voluntarily, and manifested no gastric disturbance. His bowels were moved during the day. Nothing further of interest occurred till about 3 o'clock on the following morning, when the night-nurse sent me a report that the patient had suddenly become worse, and was apparently dying. Death occurred about twenty minutes afterward.

The following is abstracted from the autopsy records: The autopsy was made eight hours after death. The pia mater was engorged with blood, but not adherent to the brain-surface. The brain-mass, as a whole, was of greatly diminished consistence, and on section showed marked congestion, although very little blood exuded from the puncta vasculosa, which were very numerous. The softening seemed general, but was most marked in the frontal and parietal segments of the brain.

On laying open the circle of Willis three thrombi were found, one in the basilar artery, successfully occluding its caliber; this thrombus was formed apparently from a thrombotic shred which had been thrown into several folds by the blood-stream, and had received some recent additions in the way of dark clotted blood. The two thrombi in the middle cerebrals were much smaller, the one on the right side being the larger of the two, but neither successfully occluded the caliber of the vessel.

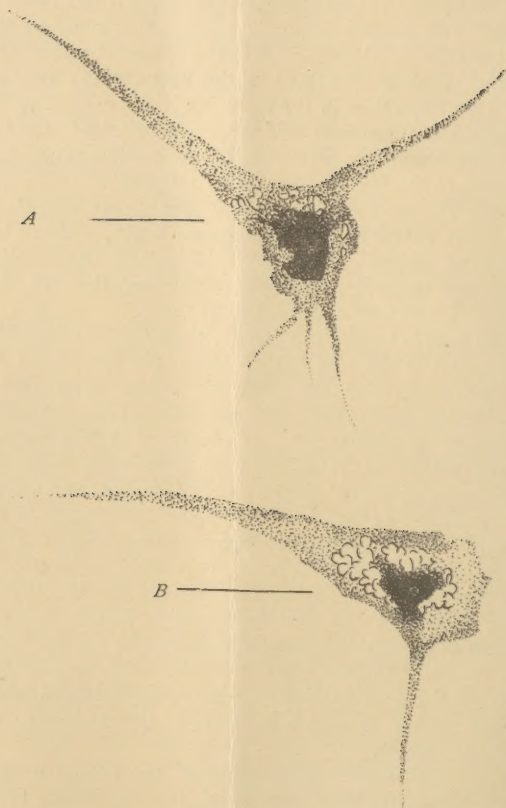
The longitudinal sinus contained an ante-mortem clot, which did not, however, occlude the caliber of the sinus. The heart contained a large thrombus in the right auricle, firmly attached to the tricuspid valve, and occupying about one-third of the space in the auricle; a smaller thrombus in the right ventricle, extending up through the pulmonary orifice; a similar clot in the left ventricle, extending up through the aortic orifice; and also one in the left auricle, firmly attached to the mitral valve. The stomach presented three ulcers, one along the lesser curvature, of very recent origin, midway between the pyloric and cardiac orifices, and two smaller ones near the pyloric orifice, each about the size of a dime. These were apparently the older.

This case is interesting from a pathologic standpoint, on account of the unusual lesions found to account for the symptoms and the patient's unlooked-for death. Considering the man's age and his previous healthy condition, it seems not improbable that his sudden manifestations of violence were coincident with the occurrence of partial thrombosis of the basilar artery, the mania being due to the serious disturbance in the cerebral circulation, and consequent impairment of nutrition in the ganglion-cells of the cortex. The circulation was still more seriously impaired when the thrombotic shreds in the middle cerebral vessels became thrown into folds and seriously narrowed the lumen of the vessels, and death was to have been expected as soon as the cerebral blood-supply became sufficiently limited by the gradually increasing obstruction to the vascular stream.

The microscopic appearances of the cortex were of interest, because they were unlike the conditions found in three cases of acute delirious mania examined previously, and all of which presented the same lesion.

The protoplasm of the cell in the neighborhood of the nucleus was almost invariably vacuolated, the de-

FIG. 1.



A. Vacuolation of protoplasm of cell; nucleus hazy in outline (upper portion). *B.* Marked vacuolation of protoplasm of cell; nucleus small and hazy in outline. (Zeiss $\frac{1}{12}$ imm. obj., Abbe camera lucida.)

gree of vacuolation varying from two or three small, rounded, light areas to large clumps of these (Fig. 1),

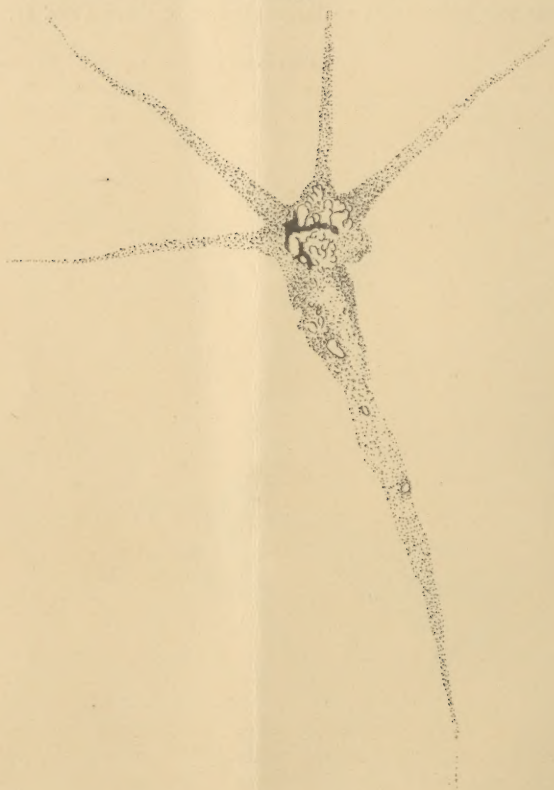
FIG. 2.



Vacuolation of protoplasm of cell and nucleus. (Zeiss $\frac{1}{2}$ imm. obj., Abbe camera lucida.)

which in many instances seriously invaded the nucleus, and in a few almost entirely replaced it. (Figs. 2 and

FIG. 3.

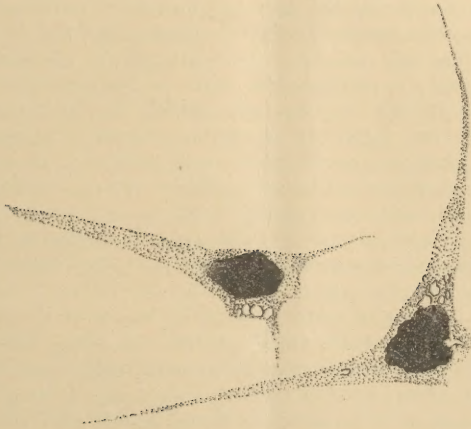


Nucleus almost entirely replaced by vacuoles. (Zeiss $\frac{1}{2}$ imm. obj., Abbe camera lucida.)

3.) The changes in the nucleus were quite extensive, but not general; when present they were quite well

marked. In very many instances the nucleus was apparently swollen; in these cases it was always very irregular in outline, but the edges were sharply defined. (Fig. 4.) In other instances portions of the circumference of the nucleus were sharply defined, while other portions were very hazy, shading off gradually into the protoplasm of the cell (Fig. 1, B). In many other in-

FIG. 4.



Enlarged and irregular nuclei. (Zeiss $\frac{1}{2}$ imm. obj., Abbe camera lucida.)

stances the nucleus was seriously encroached upon by the morbid process in the protoplasm (Fig. 2); in a few instances being occupied by one or two vacuoles, while in a few others it seemed almost entirely destroyed (Fig. 3).

The microscopic appearances are of especial interest when considered in connection with three other cases of acute delirium that have come to autopsy in this

institution in the past year and a half, because of the entirely different nature of the predominating microscopic lesion. In these latter cases pigmentary degeneration of the ganglion-cells was the most prominent feature, being widely distributed and extremely well marked. In the case under consideration there was not the slightest trace of pigmentary degeneration anywhere in the cortex; in fact, the normal deposit of pigment seemed absent from most of the cells.

In considering the apparent variability in the pathologic phenomena presented by these cases, I must confess that the cause for such variability is not entirely apparent; nevertheless we have to consider the fact that while the case was undoubtedly one of acute delirium, the nature of the delirium differed somewhat from that of cases ordinarily seen, in that it showed a marked disposition to be transient. We must also bear in mind the lesions existing in the vessels at the base of the brain as having a modifying influence on both symptoms and pathology, no such factor figuring in any of the previous cases.

In connection with this case the work of Dr. H. C. Hodge, of the Clark University, is of exceeding interest. In his study of the changes in nerve-cells after functional activity he demonstrated vacuolation of considerable extent as occurring in the nerve-cell after normal fatigue, together with considerable shrinking of the nucleus. In this case there can be little doubt of the existence of extreme fatigue, as the patient was inclined to violence, and often made night and day hideous to those with whom he was associated. Bearing this in mind, the question of the pathologic significance of the phenomena under consideration is an interesting one.

Are vacuoles in nerve-cells of any pathologic import, or is their presence simply an indication of the expenditure of nervous energy? As a partial answer to this question we should bear in mind the occurrence of the

most extreme degrees of vacuolation of nucleus and cell-body in primary and terminal dementia, forms of insanity uncharacterized by mental or physical activity of any description. The same might be said to a lesser degree of the different forms of melancholia. On the other hand, the occurrence of vacuolation in epileptic and some other forms of insanity is well known, and according to Hodge it occurs also as a direct result of physiologic fatigue.

In mental disease the nucleus is most often the seat of the morbid process, while in physiologic fatigue the protoplasm of the cell-body is chiefly affected. The forms of vacuolation also differ widely. In the conditions studied by Hodge the vacuoles seemed evenly distributed through the nerve-cell, while in the case under consideration the vacuoles were grouped together in masses, in the neighborhood of the nucleus, and in some instances involved the nucleus also. It would seem, therefore, that we may consider cellular vacuolation, aside from any physiologic process, as a pathologic factor in insanity of some importance.

In conclusion, the proposition may be made that interference with the cerebral circulation may give rise to maniacal outbursts as a direct result of nutritional disturbances in the ganglion-cells of the cortex. Nutritional disturbance is manifested, first, in the brain, by the marked diminution in the consistence of the cerebral mass, and, second, in the ganglion-cells, by vacuolation of the protoplasm of the cell-body, and in fewer instances of the nucleus also.

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